On the global spreading of toxic dinoflagellates

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Toxic dinoflagellate blooms, in a strict sense, are completely natural phenomena which have occurred throughout recorded history. For example, Captain Vancouver lost one of his crew in British Columbia in 1793 after eating contaminated mussels (paralytic shellfish poisoning), and Captain Cook is claimed to have suffered from ciguatera fish poisoning when visiting New Caledonia in 1774.

However, in the past two decades the public health and economic impacts of such events appear to have increased in frequency, intensity and geographic distribution. One example, the increased global distribution of paralytic shellfish poisoning (PSP), is illustrated in Figure 1.

Until 1970, toxic dinoflagellate blooms of *Alexandrium (Gonyaulax) tamarense* and *Alexandrium (Gonyaulax) catenella* were only known from temperate waters of Europe, North America and Japan. By 1990, this phenomenon was well documented from throughout the Southern Hemisphere, in South Africa, Australia, India, Thailand, Brunei, Sabah, the Philippines and Papua New Guinea.

Other species of the dinoflagellate genus *Alexandrium*, such as *A. minutum*, as well as the unrelated dinoflagellates *Gymnodinium catenatum* and *Pyrodinium bahamense* var. *compressum* (all species not previously known to be toxic) have now also been implicated*****. To some extent, this increased global distribution of PSP simply reflects our increased awareness of toxic species and the enormous expansion in aquaculture efforts.

Evidence is accumulating, however, that human activities contribute significantly to this increase through the stimulation of dinoflagellate blooms by cultural eutrophication and by the spreading of nuisance organisms in ships' ballast water. Cargo vessel ballast water was first suggested as a vector in the dispersal of non-indigenous marine plankton some 90 years ago. The diatom *Odontella (Biddulphia) sinensis*, well known from the tropical and subtropical coasts of the Indo-Pacific, had not been reported in European waters until 1903 when it produced dense plankton blooms in the North Sea. Since it appeared unlikely that this large diatom could have been overlooked previously and impossible that it could have been carried by currents from distant oceans, Ostenfeld (1908) suggested that this species was introduced via the water or sediment contained in ships' ballast tanks. Subsequently, Hallegraeff and co-workers (1990) confirmed this possibility by culturing the related diatom species *Odontella aurita* from a ballast water sample collected at the end of a voyage from Japan to Australia. Whereas the introduction of *O. sinensis* was apparently without harmful effects, the more recent introduction into the North Sea of the diatom *Coscinodiscus wailesii* has caused problems due to the clogging of fishing nets by extensive diatom mucous production.

The issue of ballast water transport of plankton species gained considerable interest in recent years, when Hallegraeff and co-workers (1988, 1991, 1992) brought forward evidence that non-indigenous toxic dinoflagellate species had been introduced into Australian waters in sensitive aquaculture areas, with disastrous consequences for commercial shellfish farm operations.

Paralytic shellfish poisoning was unknown from the Australian region until the 1980s, when the first outbreaks appeared in the ports of Hobart (*Gymnodinium catenatum*), Melbourne (*Alexandrium catenella*) and Adelaide (*Alexandrium minutum*). While the plankton stages of diatoms and dinoflagellates show only limited survival during the voyage in dark ballast tanks, their resistant resting spores are well suited to survive these conditions. One single ballast tank thus was estimated to contain more than 300 million toxic dinoflagellate cysts which could be germinated into confirmed toxic cultures (Hallegraeff and Bolch, 1991).

In Hobart, Tasmania, an examination of historic plankton samples, cyst surveys in sediment depth cores and genetic studies using enzyme electrophoresis and sexual compatibility experiments have provided strong circumstantial evidence that the toxic dinoflagellate *G. catenatum* was introduced in the last 10 to 20 years. Resting spores of this species have been confirmed in 4 ballast water samples entering Australian ports from either Korea or Japan. This organism has now been well-established in southern Tasmania and dense blooms in 1986, 1987 and 1991 necessitated the closure of up to 15 shellfish farms for periods up to 6 months.

Similarly, the toxic dinoflagellate *Alexandrium catenella*, which has caused the closure of shellfish farms in Port Phillip Bay, Melbourne, was not known

***** Note from the editor: a case of paralytic shellfish poisoning in 1972 near Port Moresby, Papua New Guinea, caused by *P. bahamense*, is cited by J.L. McLean (1974), SPC *Information Circular* 54.

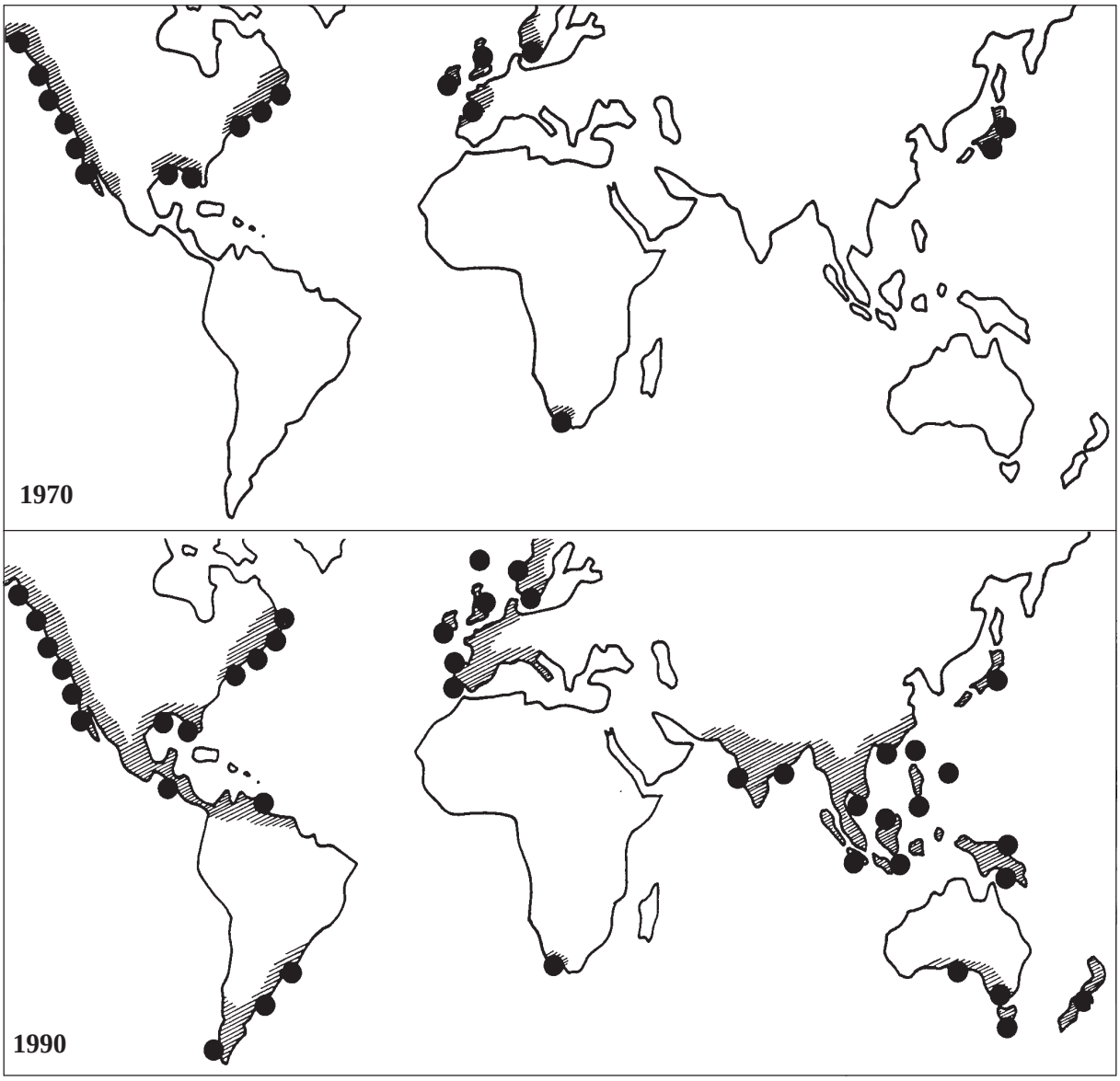

Figure 1: Global distribution of paralytic shellfish poisoning 1970 (above) and 1990 (below)

from the area before 1986. Viable resting spores of this species have been detected in ballast water being discharged into this port, and rRNA sequencing has indicated a remarkable match between ballast water and harbour water cultures of this dinoflagellate (Scholin and Anderson, 1991). Finally, the toxic dinoflagellate *Alexandrium minutum* appeared in the Port River, Adelaide, in 1986 in an area where sediment surveys carried out in 1983 failed to detect resting spores in sediments. The port of Adelaide has a shipping link with the Mediterranean, which has the only other known global population of this dinoflagellate, and rRNA sequencing has indicated a remarkable match between Australian and Spanish cultures of this species.

Another vector for the dispersal of toxic dinoflagellates (especially their resting spores) is shellfish stocks transferred from one area to another, as the faeces and digestive tracts of bivalves can at times be loaded with viable dinoflagellate cells. The Japanese seaweeds *Sargassum muticum*, *Undaria pinnatifida* and *Laminaria japonica* thus are thought to have been introduced into European waters via sporophyte stages associated with introduced Japanese oyster spat. While benthic dinoflagellates such as *Gambierdiscus toxicus* are not known to produce resistant resting spores, these species are well capable to survive dispersal as epiphytes attached to drifting macroalgae ('rafting').

Bomber and co-workers (1988) observed *G. toxicus* cells among 30 out of 198 drift algal samples collected in the Florida straits and Bahamian waters. Translocation of toxic strains of this species by ships' ballast water or as epiphytes on seaweeds fouling the hulls of ships are other vectors for the introduction of *G. toxicus* into tropical regions which may previously have been free of ciguatera (as may have occurred, for example, at Hao Atoll).

Further reading

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Ciguatera research at the Queensland University of Technology

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An active ciguatera research group has existed at the Queensland University of Technology (QUT) since 1985. This group has worked on various aspects of ciguatera research including the effects of ciguatoxin on vertebrate nerves, the symptomatology of ciguatoxin in humans and the response of fish to ciguatoxin. The group has been led by myself (M. Capra) and a medical colleague (J. Cameron) from the Princess Alexandra Hospital in Brisbane.

Since 1985 three students have completed and been awarded higher degrees for work on ciguatera research (A. Flowers and C. Blanton - Masters degrees: S. Hahn - Ph.D. degree). Currently another student (C. Purcell) is completing a Ph.D. research program. Aspects of our work on ciguatera at QUT are briefly reviewed below.

The effects of ciguatoxin on nerves

Although there have been many clinical reports describing the neurological signs and symptoms of ciguatera, very little has been documented as to the electrophysiological disturbance ciguatoxin causes in the peripheral nervous symptom.

The initial electrophysiological studies undertaken at QUT were on nerves in anaesthetised rats. The nerve chosen for study was the ventral coccygeal nerve of the rat tail. This nerve was electrically stimulated by subcutaneous needle electrodes and the elicited compound nerve action potentials were recorded by a second set of subcutaneous needle electrodes placed proximally to the stimulating electrodes. This rat tail preparation has been used to gain some insight into the mode of action of ciguatoxin on peripheral nerves.

A number of nerve conduction parameters were measured, the most useful of which were nerve conduction velocity, the duration of the refractory periods and the magnitude and duration of the supernormal period.

The refractory periods and the supernormal period give some indication of fundamental ionic and molecular processes that occur during nervous transmission. When a nerve carries an impulse there is a brief period after that impulse in which the nerve is refractory (0.5 - 4 msec). In the first part of this period (absolute refractory period) the nerve cannot carry a second impulse while in the latter part of the period (relative refractory period) a greater stimulus will elicit a second impulse.

The refractory period is related to the physiological processes controlling the movement of sodium ions (Na^+) across the nerve membrane. The regulated movement of Na^+ is the basis of normal nerve function. After the refractory periods, the supernormal period (6 - 30 msec), occurs in which